

Fascia as a Sensory Organ

A Target of Myofascial Manipulation

Robert Schleip, Ph.D.

Introduction

The body-wide network of fascia is assumed to play an essential role in our posture and movement organization. It is frequently referred to as our *organ of form*.¹ Many manual therapy approaches focus treatment on fascial tissues, and these approaches claim to alter the density, tonus, viscosity, or arrangement of fascia through the application of manual pressure.²⁻⁷ It is also assumed that these changes are not merely temporary – that they last longer than a few minutes after the immediate application. The given explanations of the involved mechanisms usually refer to the ability of fascia to adapt to physical stress. How the practitioner understands the

nature of this particular responsiveness of fascia does, of course, influence the treatment.

Tissue release and lasting changes may be due to an active contribution of the central nervous system and, particularly, fascial mechanoreceptors.



Unfortunately, fasciae are often referred to in terms of their passive mechanical properties alone. For example, experts often attribute sudden “tissue melt” to **thixotropy**. However, the results of recent studies seem to indicate that the application of temporary pressure – such as the pressure applied during a myofascial release session – would be unlikely to cause permanent tissue changes via such passive effects. In contrast, recent studies do seem to imply that tissue release and lasting changes may be due to an active contribution of the **central nervous system** and, particularly, **fascial mechanoreceptors**.

Thixotropy: The Gel-to-Sol Hypothesis

Many of the current training schools of myofascial manipulation have been profoundly influenced by Ida Rolf.⁶ In her hands-on work, Rolf applied considerable manual or elbow pressure to fascial structures to change their density and arrangement. Rolf proposed the theory that connective tissue is a *colloid substance* in which the ground substance can be influenced by the application of energy – heat or mechanical pressure – to change its aggregate form from a more dense “gel” state to a more fluid “sol” state. Typical examples of this are common gelatin or butter, which get softer with heat or mechanical pressure. This gel-to-sol transformation – also called **thixotropy**⁸ – has, in fact, been demonstrated in

connective tissues as a result of the application of long-term mechanical stress.⁹

However, the question arises: Is this model also useful in explaining the immediate *short-term* plasticity of fascia? In other words, what actually happens when a myofascial practitioner claims to feel a tissue release beneath the working hand? In most systems of myofascial manipulation, the duration of a particular “stroke” on a particular spot of tissue ranges from a few seconds to two minutes. However, practitioners often report sensing a palpable tissue release within a particular “stroke.”

Such rapid tissue transformation – less than two minutes – appears to be more difficult to explain with the thixotropy theory. As will be specified later, studies on the subject of “time and force dependency” of connective tissue plasticity suggest that either much longer amounts of time or significantly more force are required for permanent deformation of dense connective tissues.¹⁰

Furthermore, the problem of reversibility arises. In colloidal substances, the thixotropic softening effect lasts only as long as the pressure or heat is applied. Within minutes after the heat or force application, the substance returns to its state of previous rigidity – think of the butter in the kitchen. The temporary nature of the achieved tissue softening is, therefore, not an attractive implication of this theory for the practitioner.

Piezoelectricity: Fascia as a Liquid Crystal

James Oschman and others have added **piezoelectricity** as an intriguing explanation for fascial plasticity.^{11,12} When an outside mechanical pressure temporarily separates the electric centers of neutrality on the inside of a crystal lattice, a small electric charge can be detected on the crystal’s surface. This effect is called piezoelectricity. Since connective tissue can be seen to behave like a “liquid crystal”¹³ these authors propose that the cells that produce and digest collagen fibers (**fibroblasts**) might be responsive to such electric charges. To put it simply: Pressure from the outside creates a higher electric charge, which stimu-

lates the fibroblasts to alter their metabolic activity in that area.

However, the processes involved seem to require time as an important factor. The half-life span of non-traumatized collagen has been reported to be 300 to 500 days, and that of ground substance 1.7 to seven days.³ While it is definitely conceivable that the production of both materials could be influenced by piezoelectricity, both life cycles appear too slow to account for immediate tissue changes that are significant enough to be palpated by the working practitioner.

Fascial Plasticity: Traditional Explanations Insufficient

Both models, thixotropy and **piezoelectricity**, are appealing explanations for long-term tissue changes. Nevertheless, it seems they are not sufficient to account for the short-term plasticity of fascial tissues. Laboratory studies on the subject of time- and force-dependency of connective tissue plasticity (in vitro, as well as in vivo) have shown the results outlined below. In order to achieve a plastic elongation of dense connective tissues, one needs to apply:

- a. either an extremely forceful stretch of three to eight percent fiber elongation, which will result in tissue tearing, inflammation, and other side effects that are usually seen as undesirable in a myofascial session. As an example, for an 18-millimeter-wide distal iliotibial band, such permanent elongation starts at 60 kilograms (kg).¹⁴
- b. or it takes more than an hour (which can be taken at several intervals) with a less brutal one

to 1.5 percent fiber elongation, if one wants to achieve permanent deformation without tearing and inflammation.^{10,14}

Figure 1 illustrates the typical relationships for short-term strain applications. Microfailure is seen as the breaking of some individual collagen fibers and some fiber bundles, resulting in a permanent (plastic) elongation of the tissue structure. This is usually followed by a cycle of tissue inflammation and repair. Based on measurements with different kinds of paraspinal tissues, A. Joseph Threlkeld, a professor of physical therapy, calculates that microfailure of most dense connective tissues occurs at around 224 to 1,136 N, which equals 24 to 115 kg.¹⁴ While high velocity thrust techniques might create forces within that range, it seems clear that the slower soft tissue manipulation techniques are hardly strong enough to create the described tissue response.

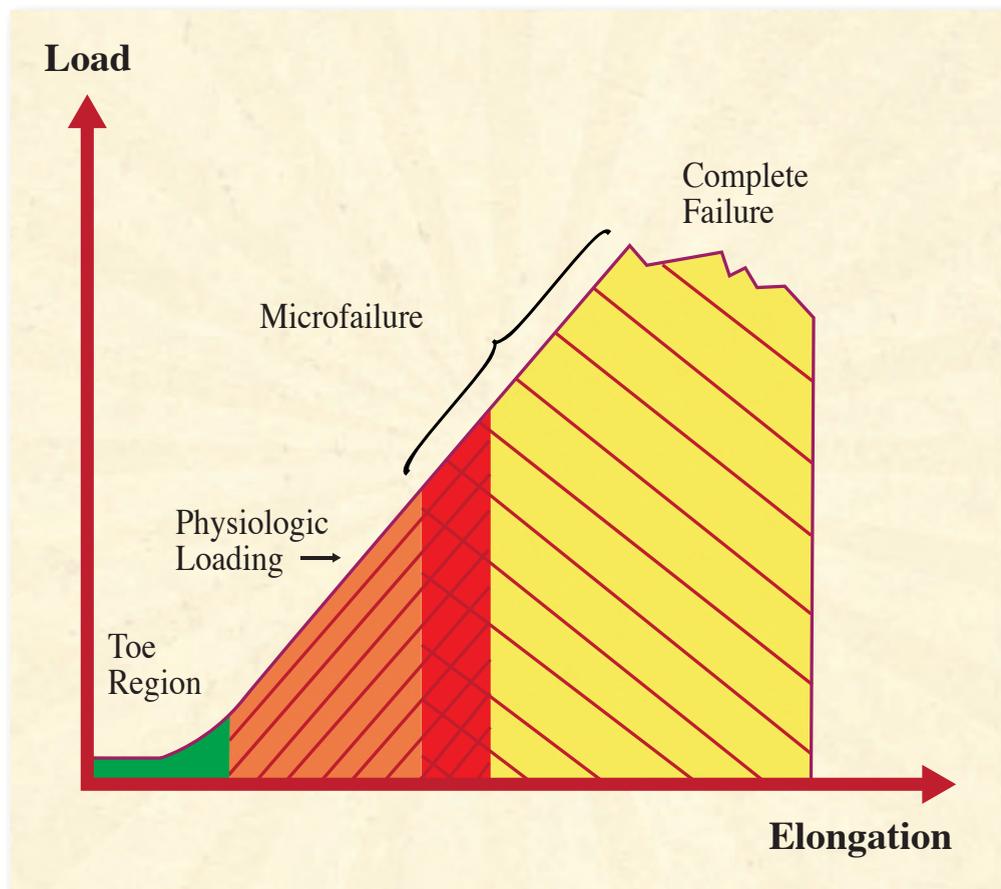


Figure 1: The stress-strain curve of dense connective tissue is illustrated here. Most forces generated during daily life load the tissue in the linear region of the curve and do not produce any permanent elongation. Microfailure with permanent elongation happens at extreme loads only and is accompanied by tearing and inflammation. The region of overlap of the microfailure zone with the physiologic loading zone varies with the density and composition of the tissue, yet for most dense connective tissues, it would be well above a 20 kilogram loading.¹⁴

Recently, these propositions have been supported by studies from biomedical engineering research professor Hans Chaudhry and his team, demonstrating that the “palpable sensations of tissue release that are often reported by osteopathic physicians and other manual therapists cannot be due to deformations produced in the firm tissues of plantar fascia and fascia lata. However, palpable tissue release could result from deformation in softer tissue such as the superficial nasal fascia.”¹⁵

In other words, while it may be possible to induce lasting changes in very soft connective tissues, such as in the areolar layer of the subcutis, the force/time dimensions used in myofascial release sessions are hardly sufficient to induce sustaining deformations in dense fascial sheets, which are usually the main focus of myofascial release sessions.

A simple thought experiment may be used for corroboration. During everyday life, the body is often exposed to loading magnitudes similar to the manual pressures in a myofascial treatment session. While body structure may adapt to long-term furniture use, it is almost impossible to conceive that adaptations could occur so rapidly that any uneven load distribution in sitting – for example, while reading this book – would permanently alter the shape of your pelvis within a minute. It seems essential, therefore, that we find additional explanations – besides the thixotropic and piezoelectric ones – to account for the palpable tissue changes that occur in a treatment session.

The Nervous System as a Rapid Self-Regulatory System

From an evolutionary perspective, it makes sense that animals have a slowly adapting plasticity system in order to adjust to patterns of long-term use. In addition to this capacity, they have also developed a more rapid system of adapting their form and local tissue density to temporary demands. Such a rapidly adapting system is likely to play a key role in the lasting tissue changes reported by manual therapists. The question then becomes: Which systems allow for this rapid adaptation?

From observation, we know that an animal's rapid regulation system is capable of adapting to how the animal **perceives its interaction** with the environment. The author's own experiments in treating anaesthetized people – which can give very similar results as manually treating fresh pieces of raw meat – have shown that myofascial release work does not affect tissues in the same way as it would if the person possessed intact neural regulation.¹⁶

Therefore, it seems plausible that this ability to quickly adapt to low-force manual manipulation is mediated by a finely tuned coordination system that is involved in the perception of our environment, as well as in sensing our own internal needs at any time. Traditionally, this body system has been called the nervous system.

The inclusion of the nervous system in attempting to understand fascial responsiveness is hardly a new concept altogether. Andrew Taylor Still, the founder of osteopathy, wrote more than a century ago:

*The soul of man with all the streams of pure living water seems to dwell in the fascia of his body. When you deal with the fascia, you deal and do business with the branch offices of the brain, and under the general corporation law, the same as the brain itself, and why not treat it with the same degree of respect.*¹⁷

Even so, many people discount the nervous system in the discussion of fascial responsiveness, because they consider the human nervous system to be orga-

nized like an old-fashioned telephone switchboard from the industrial age. Such a system would then, of course, be incapable of representing finer and more complex processes such as “life energy,” intuitive insights, rapid movement refinements, or human empathy.

The reader is cordially invited to consider this to be an outdated model. Current concepts in neurobiology primarily view the brain as a **liquid system**, in which fluid dynamics of a multitude of liquid and even gaseous neurotransmitters have come to the forefront.

It is a common opinion in the field that fascia must contain a different and much faster communication system than the nervous system. Those who hold this view often believe that the nervous system cannot adapt quickly enough for the rapid responses in

human behavior. Thus, the fascia must have a separate, faster adaptation system. However, transmission of impulses in our nervous system often happens via messenger substances that travel along neural pathways, as well as through the blood, lymph, cerebrospinal fluid, or ground substance.¹⁸

This global system for *rapid* body regulation is inseparably connected to the endocrinal and immune systems, and it also works with complex feedforward system dynamics. Rather than picturing the nervous system as a hard-wired electric cable system – which in the view of many bodyworkers is then, of course, incapable of being involved in more subtle energetic phenomena – picture it in your mind's eye as a *wet tropical jungle*.¹⁹

This jungle is a self-regulatory field – involving complex feedforward system dynamics – with an amazing amount of complexity, plasticity, and continuous reorganization. Such a complex neural field could easily be involved in the rapid fascial changes experienced during manual therapy.

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The Nervous and Fascial Systems are Inseparably Interwoven

Our central nervous system receives its greatest amount of sensory input from myofascial tissues. Unfortunately, the neural dynamics of fascia have rarely been explored in detail. The majority of these sensory neurons within myofascial tissues are so small and complex that, until recently, little has been known about them.²⁰

If we discover exactly how the nervous system connects with fascia, we will be in a better position to uncover the causes of patients' myofascial release during passive bodywork sessions.

Fascia contains four types of sensory nerve endings, which are responsive to mechanical stimulation:

1. **Golgi organs**
2. **Ruffini receptors**
3. **Pacini corpuscles**
4. **Interstitial receptors**

Together, these sensory nerve endings can be called **fascial mechanoreceptors**, which means that they respond to mechanical tension and/or pressure. Such mechanoreceptors have been found in intramuscular, as well as extramuscular, fascial tissues.

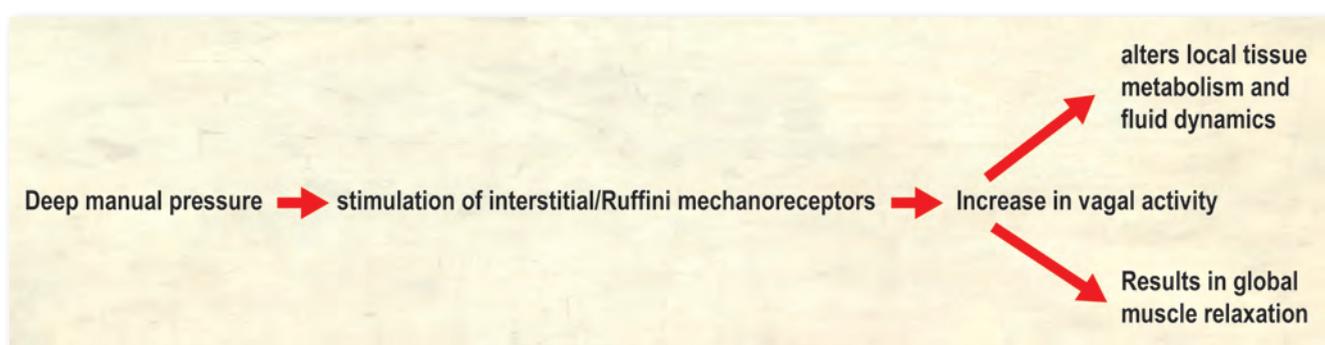
Research has shown that stimulation of fascial mechanoreceptors triggers changes in the nervous system, as well as global muscle relaxation. For example, it has been demonstrated that deep mechanical pressure to the human abdominal region²¹ or sustained

pressure to the pelvis²² produce parasympathetic reflex responses,^a including synchronous cortical EEG patterns, **increased vagal activity** (which causes a decrease in heart rate), and decreased EMG activity.

According to the model of hypothalamic tuning states, developed by Ernst Gellhorn, this increase in vagal tone not only triggers **autonomic nervous system changes**^b and related inner organs, but it also tends to activate the anterior lobe of the hypothalamus. Such a “trophotropic tuning” of the hypothalamus then induces more quiet emotional activity, an increase in synchronous cortical activity, and a **lower overall muscle tonus**, or softening of the muscles.²³

In other words, deep mechanical pressure to these areas causes a person to be more relaxed. Therefore, it appears that deep manual pressure – specifically if it is slow or steady – stimulates particular fascial mechanoreceptors, which results in an increase of vagal activity.

As described later, the most likely candidates for such receptors are the Ruffini endings, as well as interstitial receptors in fascial tissues. This increase in vagal activity then not only induces an alteration in local fluid dynamics and tissue metabolism, but it also results in **global muscle relaxation**, as well as a more peaceful mind and less emotional arousal.



a The parasympathetic nervous system is the system typically associated with rest and relaxation.

b Examples of autonomic nervous system changes include changes in heart rate, blood pressure, respiration, and so on.

In addition, mechanoreceptors are also found abundantly in visceral ligaments, as well as in the dura mater of the spinal cord and cranium. It, therefore, seems quite plausible that many of the benefits of osteopathy on these areas could be sufficiently explained by

a stimulation of mechanoreceptors. Such effects can explain profound autonomic changes without the need to rely on more esoteric assumptions, such as a breathing cerebrospinal liquid system.²⁴

Fascial Mechanoreceptors Linked with Endocrine System

Fascial mechanoreceptors are not only intricately interwoven with the autonomic nervous system, but they are also vital to communication within the endocrine system. For example, many of the sensory neurons of the enteric brain^c are mechanoreceptors, which – if activated – trigger, among other responses, important neuroendocrine changes. These include a change in the production of serotonin – an important cortical neurotransmitter – as well as other neuropeptides, such as the substance histamine, which increases inflammatory processes.

Obviously, fascia are not the passive players we often envision them to be – responding

to our manual manipulations as simply as a lump of molding clay. Their close-knit involvement with both the nervous and endocrine systems means a bodyworker should approach fascial manipulation with a deeper understanding of the dynamic properties of fascial innervation.

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The more we understand about the communication between fascial mechanoreceptors and the body's systems, the more likely we are to create optimal manual therapy sessions for our clients. The following discussion delves more deeply into the relationship between the nervous system, the endocrine system, and fascial mechanoreceptors.

^c Recent discoveries concerning the richness of the enteric nervous system²⁵ have taught us that our “belly brain” contains more than 100 million neurons and works largely independently of the cortical brain. It is interesting to note that the limited connection between these two brains – only a few thousand neurons – consists of nine times as many neurons involved in processes in which the lower brain tells the upper one what to do, compared with the number of neurons involved in the top-down direction.

Fascial Mechanoreceptor 1: The Golgi Receptors

In his book *Healing Through Touch*, John T. Cottingham presented a milestone proposal when he suggested the first neurophysiological concept for myofascial bodywork.²⁶ This concept was readily adopted by other authors^{7,16} and will be recapitulated here. Golgi receptors are said to be ubiquitous in dense connective tissues. They exist in ligaments, as *Golgi end organs*, in joint capsules, and around myotendinous junctions, as *Golgi tendon organs*.

These sensory receptors respond to slow stretch by influencing associated alpha motor neurons, via the spinal cord, to lower their firing rates – to decrease the active muscle tone in related muscle fibers. Cottingham suggested that during soft tissue manipulation – as well as in Hatha yoga postures and slow active stretching – these Golgi receptors are stimulated, resulting in a lowered firing rate of specific alpha motor neurons, which translates into a tonus decrease, or softening, of the related tissues.

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The Effect of Stimulating Golgi Organs

Unfortunately, the experimental research on Golgi tendon organs suggests that passive stretching of a myofascial tissue – with the muscular fibers being in a relaxed state – does *not* stimulate these tendon receptors.²⁷ These experiments, usually performed on laboratory animals, propose that such stimulation of the tendon receptors happens only when the muscle fibers are actively contracting.

The reason for this lies in the arrangement of the Golgi tendon receptors. They are arranged in series with the muscle fibers, with the tendon having a much higher stiffness than relaxed muscle fibers. When the muscle is passively elongated, most of the stretch will be taken up, or “swallowed,” by a resulting elastic elongation of the muscle fibers.^d

This is, of course, different in active client contractions, in which contractile muscle fibers acquire an increased stiffness, and the Golgi tendon organs function to provide feedback information about dynamic force changes during the contraction.²⁸

Does this mean deep tissue work – in which the client is often passive – will not involve the Golgi reflex loop? Perhaps, but not necessarily. The *in vitro* studies discussed above were conducted using preparations in which muscles were surgically isolated – freed from their lateral myofascial adherences to surrounding structures. However, as shown by the extensive work of Peter Huijing and others, intact muscle tissues exhibit very different force transmission dynamics than isolated muscles.²⁹

Furthermore, it is important to note that *fewer than 10 percent* of the Golgi receptors are found wholly within tendon – and the studies described above^{27,28} were performed *only* on Golgi tendon

receptors. The remaining 90 percent of the Golgi receptors are located in the muscular portions of myotendinous junctions, in the attachment transitions of aponeuroses, in capsules, and in ligaments of peripheral joints.³⁰

Taking these considerations into account, it cannot be excluded that passive tissue stretch may be able to stimulate some Golgi receptors, particularly if the tissue is stretched in directions **other than along the main muscle-tendon axis**. In addition, if one applies **strong local pressure** to the fascial connection, the relatively intense, direct pressure may cause Golgi receptors to be activated. However, the chance of eliciting such responses appears to be higher if the related muscle fibers are not in a state of complete relaxation.

^d For clarification, the following analogy may be helpful. When a tight rope and a soft noodle are serially connected (one after the other in one straight continuous line), a pull on both of them will hardly result in any extension of the rope.

Fascial Mechanoreceptors 2 and 3: Ruffini and Pacini Corpuscles

Both the Pacinian/Paciniform and the Ruffini bodies are found in all types of dense connective tissue – muscle fasciae, tendons, ligaments, aponeuroses, and joint capsules. In myotendinous junctions, the Pacinian corpuscles are more concentrated in the tendinous portion, as opposed to the Golgi tendon organs, which are more concentrated in the muscular portion.

They have also been shown to be more concentrated in the deeper portions of joint capsules; in deeper spinal ligaments; in plantar, as well as palmar tissues; in the peritoneum and enveloping muscular fasciae, such as the antebrachial, crural, and abdominal fasciae; and in the fascia of the masseter and the lateral thigh.³¹

The Ruffini endings are particularly densely distributed in tissues associated with regular stretching, such as the outer layer of joint capsules, the dura mater, the ligaments of peripheral joints, and the deep dorsal fascia of the hand. At the knee joint, the Ruffini endings are especially found at anterior and posterior ligamentous and capsular structures, whereas Pacinian bodies are more accumulated medially and laterally of the joint.³²

In addition, an immunohistochemical examination of the human thoracolumbar fascia revealed that it is richly populated by such mechanoreceptors.³³ In this study, researchers found a high presence of Pacini and Ruffini endings, but no Golgi receptors.

The Effect of Stimulating Pacini Receptors

The group of Pacini receptors consists of the large Pacini corpuscles and the slightly smaller *Paciniform* corpuscles. The egg-shaped Pacini bodies respond to rapid changes in pressure – but not to constant unchanging pressure – and to vibration. A bit smaller are the *Paciniform* corpuscles, which have a similar function and sensitivity. Therefore, it seems likely that the Pacinian receptors are being stimulated only by **high-velocity thrust manipulations and vibratory or oscillatory techniques**.

Stimulation of Pacini receptors does not yield any clearly predictable muscle tonus increase or decrease, but it does trigger **heightened local proprioceptive attention of the central nervous system** to the stimulated fascial region.

In case of a local sensomotor amnesia, as described by Thomas Hanna,³⁴ such stimulation may, thus, have beneficial effects and may result in a refined cortical body representation and improved local neuromuscular coordination.

The Effect of Stimulating Ruffini Endings

The smaller and more longitudinal Ruffini organs do not adapt as quickly as Pacini receptors, and, therefore, respond to constant pressure. In contrast to the Pacini, the Ruffini endings are activated by **slow and deep “melting quality” soft tissue techniques, as well as faster strokes**.

Two aspects of Ruffini endings seem important:

1. They are especially responsive to **tangential forces and lateral stretch**.³⁵
2. It has been shown that stimulation of Ruffini receptors tends to induce a lowering of **sympathetic nervous system activity**.^{32,e} This seems to fit to the common clinical finding that slow deep tissue techniques tend to have a relaxing effect on local tissues, and, indeed, on the whole organism.

In order to examine the potential neural dynamics of Ruffini receptors in myofascial manipulation, let us use the following scenario as a reference point. Imagine a practitioner working slowly with the connective tissue around the lateral ankle, in an area with no striated muscle fibers. Choosing this reference scenario allows us to focus on intrafascial dynamics only and – for the purpose of this chapter – to ignore the stimulation of intramuscular mechanoreceptors and other effects that would be involved in the analysis of many other myofascial working situations.

If the practitioner in this scenario reports a “tissue release,” what has happened? Possibly, the manual touch stimulated some Ruffini endings, which, in turn, triggered the central nervous system to change the tonus of some motor units in the muscle tissue that was mechanically connected to the tissue beneath the practitioner’s hand (Fig. 4).

e The sympathetic nervous system is often associated with the fight or flight response. Increase in sympathetic nervous system activity results in stress. An inhibition of the sympathetic nervous system has a relaxing effect.

Fascial Mechanoreceptor 4: Interstitial Receptors

Interstitial neural fibers are often ignored, yet they account for almost 80 percent of the sensory fibers within a typical motor nerve.^f Only a small fraction of a motor nerve's sensory fibers belong to the well-known types I and II sensory fibers, which originate

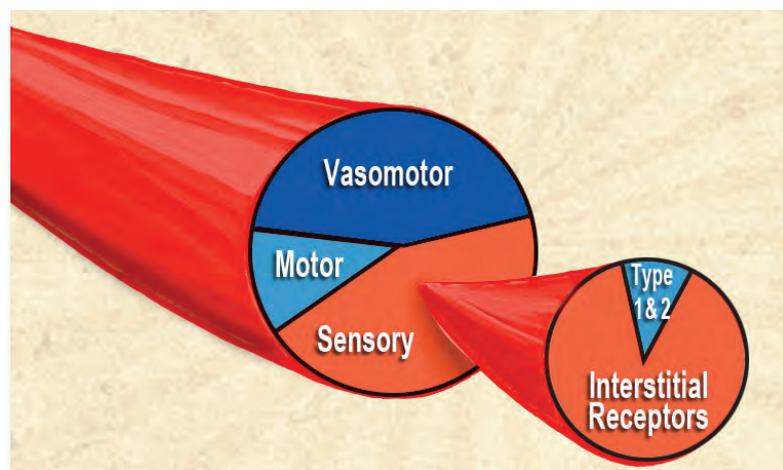


Figure 2: Within a typical muscle nerve there are almost three times as many sensory neurons than motor neurons. Note that only a small portion of the sensory information comes from the type I and II receptors originating in muscle spindles – Golgi receptors, Pacinian corpuscles, and Ruffini endings. The majority of the sensory input comes from the group of type III and IV receptors, or interstitial receptors, which are intimately linked with the autonomic nervous system.

in muscle spindles – Golgi organs, Pacini corpuscles, and Ruffini endings (Fig. 2). The majority, or four times as many, belong to an interesting group of types III and IV sensory interstitial fibers, which are hardly mentioned in most textbooks.³⁶

These hidden neurons are much smaller in diameter than the other three fascial mechanoreceptors and are now commonly called *interstitial muscle receptors*. A better name would be *interstitial myofascial tissue receptors* because they also exist abundantly in fascia. A minority of these fibers are covered by a very thin myelin sheath (type III, also called A-delta fibers), but 90 percent of them are unmyelinated (type IV or C fibers). These interstitial receptors are slower than the type I and II sensory fibers, and most of them originate in free nerve endings.

In the past, it was assumed that these nerve endings were mostly pain receptors. Some have also been shown to be involved in thermo- or chemoreception. Although many of these receptors are multimodal, research has shown that the majority of interstitial receptors do, in fact, function as mechanoreceptors.³⁶

The Effect of Stimulating Interstitial Receptors

While the type III and type IV fibers exhibit some important differences in certain physiological aspects, they do express common features regarding their mechanoreceptor functions. The large group of interstitial mechanoreceptors can be subdivided into two groups of equal size: low-threshold pressure units (LTP units) and high-threshold pressure units (HTP).

A study of cat Achilles tendon revealed that about half of the type III and IV endings encountered were LTP units and **responded to light touch**, even touch as light as “with a painter’s brush.”³⁶ Based on this finding, doesn’t it

seem possible – if not probable – that soft tissue manipulation might involve stimulation of these type III and type IV receptors?

This raises the question of the natural functional role of interstitial mechanoreceptors in the body. What regular consequences or reactions have been associated with an excitation of this hidden and rich sensory network? Of course, some of them function as **pain receptors**. However, research conducted as early as 1974 revealed that the type III and type IV receptors in the fasciae of the temporalis, masseter, and infrahyoid muscles show “responses to the mandibular movement and the **stretching of the fascia and the skin.**”³⁷

Thus, the study suggested that these nerve

^f Although many of the nerve fibers in a typical motor nerve have a vasomotor function, regulating blood flow, the largest group of fibers is sensory. If one studies a typical muscle nerve, such as the tibial nerve, it consists of almost three times more sensory fibers than motor fibers. This points to a fascinating principle – that sensory refinement seems to be much more important than motor organization.

endings are concerned “with the sensation of position and movement of the mandible. It is, therefore, quite likely that some of these receptors may also be responsive to the tissue deformations achieved in therapeutic manual fascia manipulations.

Furthermore, the majority of these type III and type IV mechanoreceptors have been shown to have **autonomic functions**. In other words, stimulation of their sensory endings leads to a change in heart rate, blood pressure, respiration, and so on.

Stimulation of type IV receptors tends to increase arterial blood pressure,³⁸ whereas stimulation of type III receptors can both increase *and* decrease blood pressure. Several studies have shown that an increase of static pressure on muscles tends to lower arterial blood pressure.³⁶ It seems that a major function of this intricate network of interstitial tissue receptors is to **fine-tune the nervous system’s regulation of blood flow** according to local demands, and that this is done via very close connections with the autonomic nervous system.

Working with the Whole Italian Family

For a simplified analogy, Figure 3 is a cartoon version of some of the clinically important features of the four types of fascial mechanoreceptors. The bodybuilder image of Signor Golgi illustrates one proposed working style for addressing Signor Golgi, who is the oldest of the three Italian brothers. A Golgi receptor likes it if he can be muscularly active,

and he likes strong work. When he is addressed that way, he gets easy and relaxed.

His brother, Signor Pacini, on the other hand, needs constant stimulation. Like a person with attention deficit disorder, it is difficult for him to pay attention to you if your touch is too slow, steady, or monotonous. However, if you entertain him with constant

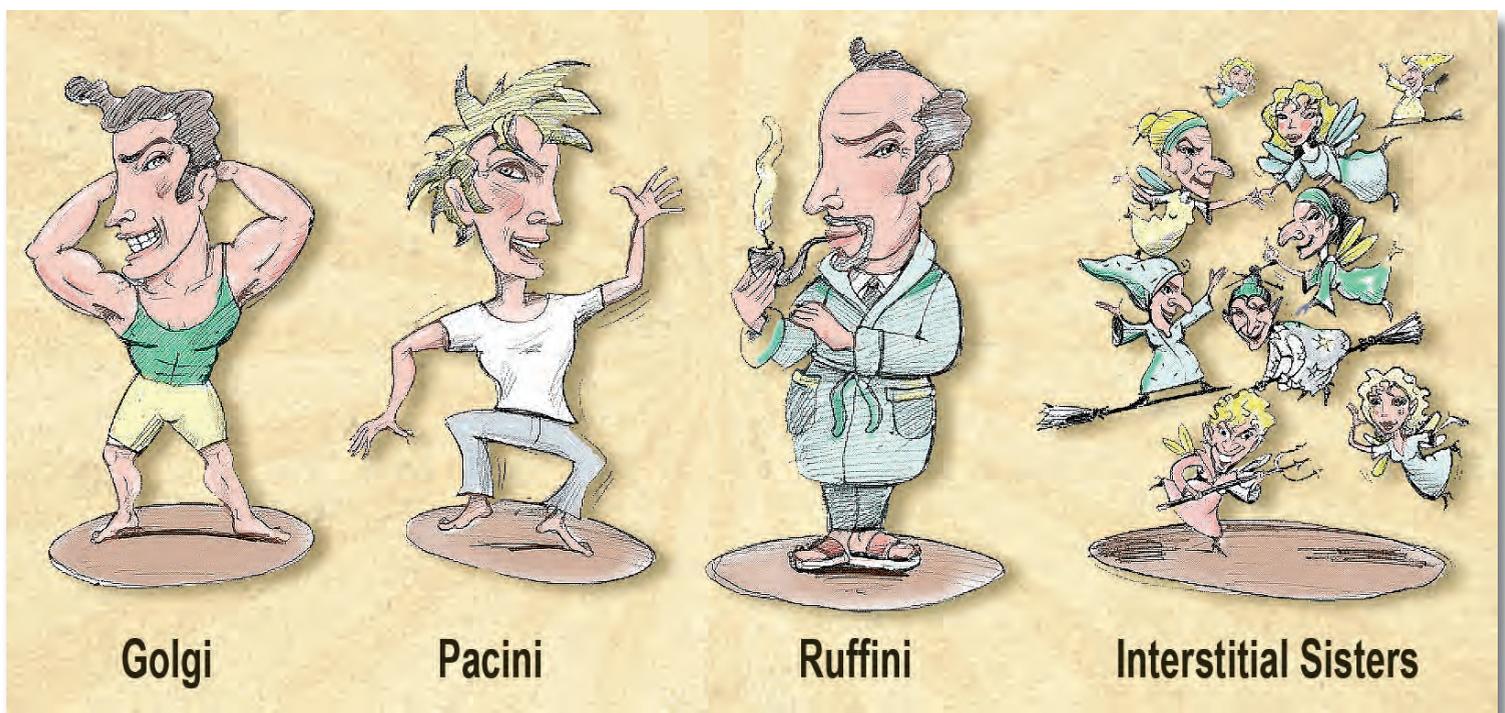


Figure 3: This is a simplified cartoon model of the four fascial mechanoreceptors. This Italian family consists of three older brothers and their little sisters. Signor Golgi prefers to be muscularly active, and he likes it strong. When addressed that way, he is a jolly good fellow. At the other end of the spectrum, his brother Signor Pacini needs constant stimulation, which he rewards with vivid attention. Signor Ruffini appreciates it when you go slowly, which he rewards by inducing a global relaxation (smoke). Finally, the numerous little sisters, representing the interstitial receptors, can have witch-like properties, as well as angel-like healing properties.

changes and stimulation, he rewards you with vivid attention.

Finally, let's look at the third of the three Italian brothers, Signor Ruffini. He is not the fast cappuccino guy, but an old-fashioned pipe smoker with a beard. Signor Ruffini likes it slow, and he prefers to address an issue at an angle, not in straightforward attacks. If you approach him in a slow manner and at the right tangential angle, he likes it a lot, and he will disperse some nice relaxing smoke that will have global effects throughout the whole body.

However, the power of the three brothers is well-matched – or even surpassed – by that of their numerous little sisters – the interstitial receptors. Some of these tiny nerve endings can have witch-like properties, evoking both temporary and long-term pain sensitization processes in their neighborhood. Others can have angel-like healing properties, if addressed in the right manner.

While many practitioners may have a favorite working style, possibly primarily affecting only one of the four described mechanoreceptors, it is advisable to address the remaining receptor types as well, at least once a while. As in family systems therapy, it is suggested that ignoring some members may be less efficient than giving at least *some* attention to them all.

Specific instructions on how to optimally address each of the depicted mechanoreceptors are best taught in the setting of a hands-on class. As an example, working approaches for addressing the Ruffini receptors are usually attractive to practitioners who already love a melting touch quality, in which the hand carefully listens to the tissue to understand the exact tangential angle the tissue responds to most. This myofascial working approach correlates well with important elements in traditional Rolfing® and also with slow-melting approaches in fascia-directed osteopathy.

Working approaches for addressing the Ruffini receptors are usually attractive to practitioners who already love a melting touch quality.

Working with the interstitial receptors in the periosteum might recall *Chua Ka*, the traditional bone massage with which Mongolian warriors reportedly freed their body from fear before going into battle. Here, the pressure or shear of the practitioner on the periosteum is slowly increased until a slight sympathetic activation, along with a minimal orienting motor response, can be noticed.

These reactions may include a slight widening of the pupils, an increase or extension of a respiratory inhalation movement, a tiny watering of the eye, a slight blushing of the face, or a minimal turn of the head toward the practitioner. However, any motor expression of a withdrawal response, no matter how small, should alert the practitioner to *back off*.

Examples of a withdrawal response include narrowing of the eyes, neck preparation to turn away from the practitioner, tightening of the lips, a more angular breathing movement of the lower belly wall, and so on. Ideally, the client supports the periosteal work with active movement participations from the inside, thereby increasing the pressure or shear at the working site in a moderated manner.

While such periosteum work should not be done on inflamed myofascial tissues or other areas with heightened pain sensitivity, it usually works quite well in those periosteal areas that are located close to pain areas, but have normal pressure sensitivity.

If preceded by respective training to extend one's tactile sensitivity into a well-crafted hand tool, some practitioners profit from using a wooden or metal tool in the hand to direct the periosteal pressure with more precision.

Fascial Mechanoreceptors: An Entry for Changing Skeletal Muscle Tone

By now, there is little doubt that myofascial manipulation involves stimulation of intrafascial mechanoreceptors. Studies support the fact that this manipulation is also an entry for changing skeletal muscle tone. For example, slow deep pressure on the soft tissue of cats has been shown to lead to a **reduction in muscle tonus** as measured by electromyogram (EMG) activity.³⁹ On the other hand, sudden deep tactile pressure, or pinching, and other types of strong and rapid manipulations have been shown to induce a general contraction of skeletal muscles.^{40,g}

The mechanism by which manual manipulation leads to muscle tonus change appears to involve the central nervous system loop (Fig. 4). The stimulation of mechanoreceptors can lead to an altered **proprioceptive input to the central nervous system**, which can easily result in a changed tonus regulation of

motor units associated with this tissue. In the case of slow deep pressure, the related mechanoreceptors are most likely the slowly adapting Ruffini endings and some of the interstitial receptors, though additional receptors might also be involved, such as spindle receptors in nearby affected muscle fibers, or possibly some intrafascial Golgi receptors.

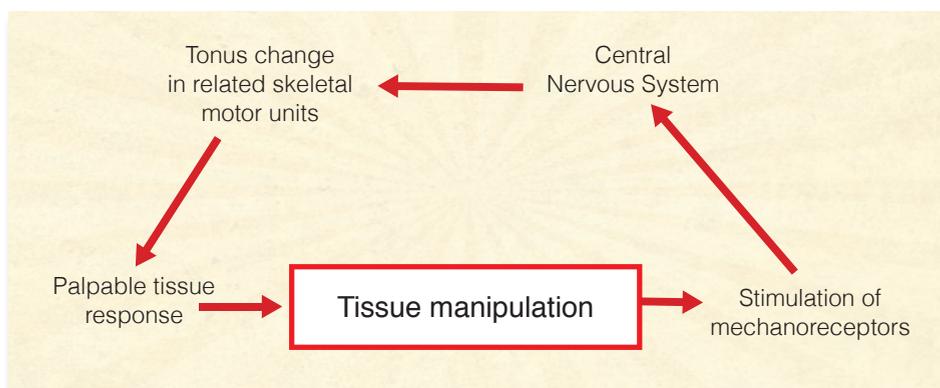


Figure 4: This shows the Central Nervous System Loop.²⁶ Stimulation of mechanoreceptors leads to a lowered tonus of skeletal motor units, which are mechanically linked to the tissue under the practitioner's hand. The involved intrafascial mechanoreceptors are most likely Ruffini endings, Pacinian corpuscles (with more rapid manipulations), some of the interstitial receptors, and possibly some intrafascial Golgi receptors.

Mechanoreceptor Stimulation and Gamma Motor Tone Regulation

Measurements of the mechanoreceptors of knee joint ligaments have shown that their stimulation leads only to weak effects in alpha motor neurons, but to powerful changes in gamma motor neurons. While the alpha and gamma motor systems are usually coactivated, there are some important differences between them.

The alpha system originates primarily in the cortex, and it is particularly involved in volitional and precise movements of the extremities. The gamma system originates in older brain stem structures and plays a strong role in the more global and unconscious postural organization of antigravity extensor muscles and chronic musculo-emotional

attitudes.^{7,42,43} Because the stimulation of mechanoreceptors in the knee joint leads to greater changes in gamma neurons than alpha neurons, these ligamentous mechanoreceptors are probably used as **proprioceptive feedback for preparatory regulation**, or preprogramming, of muscle tonus around this joint.⁴⁴

For myofascial practitioners, this finding is fascinating news, as it suggests that stimulation of fascial mechanoreceptors may lead primarily to changes in gamma motor tone regulation. This suggests that manual stimulation of ligamentous mechanoreceptors could be a potent avenue for inducing changes in subconscious muscular holding patterns.

g This is particularly shown in "genetic flexor muscles,"⁴¹ which are innervated via a ventral primary ramus from the spinal cord.

Muscles are Not Functional Units

When discussing any changes in motor organization, it is important to realize that the central nervous system does not operate “in muscles” – a muscle is never activated as a whole. The functional units of the motor system are the so-called *motor units*. There are several million of these motor units in the human body. They function much like a school of fish that have learned to swim together. Depending on the quality of sensory feedback, these millions of motor units can be individually regulated.⁴⁵

Based on this background, we can apply these details to a reference scenario in which a practitioner is working on the connective tissue around the lateral ankle. When the practitioner reports a tissue release, it may be that it is caused by a lowered firing rate of only a few fish (motor units) in the vicinity, and that this movement is transmitted to the tissue under the practitioner’s hand. If the practitioner then feels the change and responds in a supportive way toward these particular fish, other fish may soon follow the new direction, which leads to additional “release sensations” for the practitioner (Fig. 5).



Figure 5: This illustration depicts myofascial tissue as a school of fish. A practitioner working with myofascial tissue may feel several of the motor units – the fish – responding to the touch. If the practitioner responds supportively to this new behavior, the working hand will soon feel other “fish” responding. *Illustration by Twyla Weixl, Munich, Germany.*

Influencing Local Fluid Dynamics

There are, however, probably additional feedback loops involved. Let’s remember that it is the large group of interstitial receptors that makes up the majority of sensory input from myofascial tissue. Their activation triggers the autonomic nervous system to change the local pressure in fascial arterioles and capillaries.^h

According to Lawrence Kruger, researcher and distinguished professor of neurobiology at the University of California, Los Angeles, many of the interstitial fibers – if strongly stimulated – apparently can also influence **plasma extravasation** – the extrusion of plasma from blood vessels into the interstitial fluid matrix.³⁵

Such a change of local fluid dynamics means a

change in the viscosity of the extracellular matrix. This harks back to Ida Rolf’s originally proposed gel-to-sol concept,⁶ this time with the inclusion of the client’s nervous system.

The fact that interstitial fibers can influence plasma extravasation also supports Mark F. Barnes’ proposal that myofascial manipulation might involve a change of the system of ground regulation (which includes vascular pathways).^{46,i} With an increased renewal speed in the ground substance, it also appears more likely that the piezoelectric model that was explored in the early part of this chapter might play a role in immediate tissue plasticity.

^h Additionally, stimulation of Ruffini endings is reported to have a similar effect in terms of a lowering of sympathetic activity.³²

ⁱ This is defined by physician and researcher Dr. Alfred Pischinger as a functional unit of final vascular pathways, connective tissue cells, and final vegetative neurons.⁴⁷

Given that a myofascial manipulation then affects **both the local blood supply and the local tissue viscosity**, it is quite conceivable that these tissue changes could be rapid and significant enough to be felt by the listening hand of a sensitive practi-

tioner. Figure 6 illustrates this first autonomic feedback loop, called the “Intrafascial Circulation Loop,” based on the work of Dr. Jere H. Mitchell and Dr. Robert F. Schmidt.³⁶

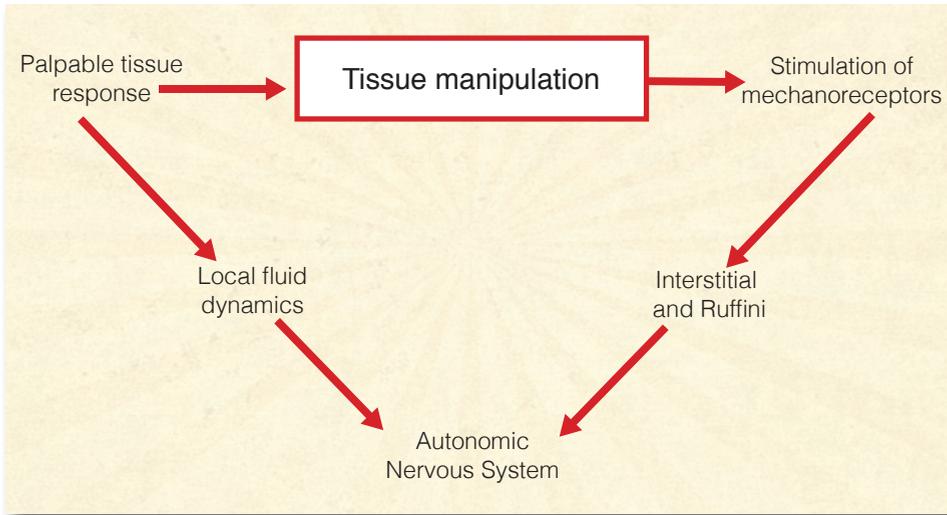


Figure 6: This illustration of the Intrafascial Circulation Loop is based on the work of Dr. Jere H. Mitchell and Dr. Robert F. Schmidt.³⁶ Fascia is densely innervated by interstitial tissue receptors. The autonomic nervous system uses the input of these receptors, along with the input of some Ruffini endings, to regulate local fluid dynamics in terms of an altered blood pressure in local arterioles and capillaries, plus plasma extravasation and local tissue viscosity. This change might then be felt by the hand of a sensitive practitioner.

Hypothalamic Tuning

In addition, there is at least one more autonomic feedback loop. Slow deep pressure usually leads to a more **parasympathetic, or relaxed, state**. This activates the more trophotropic anterior lobe of the hypothalamus to lower the overall tonus of the body musculature. According to the previously discussed

findings of Ernst Gellhorn, professor of neurophysiology at the University of Minnesota, this results in global neuromuscular, emotional, cortical, and endocrinal changes that are associated with deep and healthy relaxation.²³ This “Hypothalamus Loop” is illustrated in Figure 7.

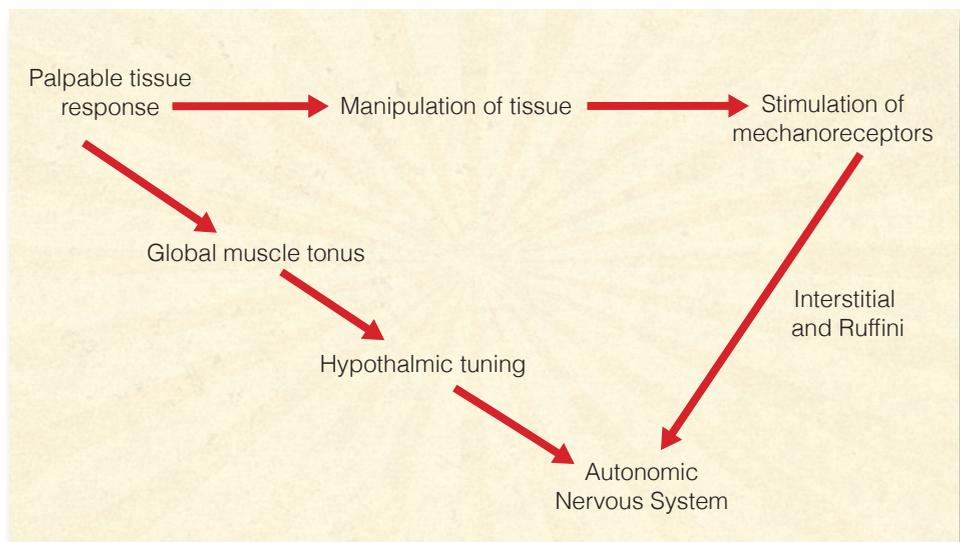


Figure 7: This figure shows the Hypothalamus Loop, based on the work of neurophysiology professor Ernst Gellhorn.²³ Slow deep pressure can stimulate interstitial mechanoreceptors, which can trigger an increase in vagal tone. This, in turn, leads toward more trophotropic tuning of the hypothalamus and, subsequently, to a reduced tonus in the body musculature.

A Brief Review

In summary of what we have learned so far, consider Table 1. We have discovered that fasciae are rich in four types of mechanoreceptors. Each type has a preferred location and is responsive to a different type of manual manipulation. When stimulated, each type

of receptor has a different effect on the body – many of these effects are made possible by the complex feedback loops previously discussed.

Table 1

Mechanoreceptors in Fascia

<i>Receptor type</i>	<i>Preferred location</i>	<i>Responsive to</i>	<i>Known results of stimulation</i>
Golgi Type Ib	<ul style="list-style-type: none"> • Myotendinous junctions • Attachment areas of aponeuroses • Ligaments of peripheral joints • Joint capsules 	<p>Muscular contraction in golgi tendon organs</p> <p>Probably to strong stretch only in other golgi receptors</p>	Tonus decrease in related striated motor fibers
Pacini and Paciniform Type II	<ul style="list-style-type: none"> • Myotendinous junctions • Deep capsular layers • Spinal ligaments • Investing muscular tissues 	Rapid pressure changes and vibrations	Proprioceptive feedback for movement control (sense of kinesthesia)
Ruffini Type II	<ul style="list-style-type: none"> • Ligaments of peripheral joints • Dura mater • Outer capsular layers and other tissues associated with regular stretching 	<p>Like Pacini, but also to sustained pressure</p> <p>Especially responsive to tangential forces (lateral stretch)</p>	Inhibition of sympathetic activity
Interstitial Types III and IV	<ul style="list-style-type: none"> • Most abundant receptor type, found almost everywhere, even inside bones • Highest density in periosteum 	<p>Rapid as well as sustained pressure changes</p> <p>(50 percent are high-threshold units, and 50 percent are low-threshold units)</p>	<p>Changes in vasodilation</p> <p>Plus, apparently in plasma extravasation</p>

Evidence that Fasciae Contain Muscle-Like Contractile Properties

We have learned that fascia is more “alive” than has previously been understood in Western medicine. This aliveness includes not only the sensory dimension, which we have recently addressed, but also a motor dimension. In other words, fascia is not an inert tissue that only responds to outside forces. From what we know today, fascia also has the ability to change its tonus autonomously, independent of outside muscular forces. Let’s explore this in more detail.

L’Hocine Yahia, director of the Bioperformance Analysis and Innovation Laboratory, and his team in Montreal – after doing a study on the sensory innervation of fascia, which we discussed earlier in this chapter – also conducted a fascinating examination on the viscoelastic properties of the lumbodorsal fascia.⁴⁸ Performing various repeated tests with dynamic and static traction loading on fresh pieces of lumbodorsal fascia from cadavers, their findings supported the well-known force- and time-dependent viscoelastic phenomena that have already been described by other researchers – creep, hysteresis, and stress relaxation.⁴⁹ However, they also described a new phenomenon for the first time, which they termed **ligament contraction**. When stretched and held at a constant length repeatedly, the tissues began to slowly increase their resistance.

Since nobody had described such spontaneous contraction of connective tissue before, they performed repeated tests involving different temperatures, solutions, and humidity, all with similar results. After very carefully ruling out the possibility of an experimental artifact, Yahia and his team finally concluded:

A possible explanation for the contraction of fascia held under isometric conditions could be the existence of cells with muscle-like contractile properties in the lumbodorsal fascia. Indeed, many visceral muscles possess the ability to contract spontaneously. Price et al (Price 1981) demonstrated that strained and isometrically held intestinal muscles undergo relaxation followed by contraction.⁵⁰ In order to test these specimens in a relaxed state

(without spontaneous contraction), they used diverse techniques to suppress spontaneous activity, amongst them the use of epinephrine. A histological study of lumbodorsal fascia would, therefore, be desirable to evaluate whether muscles play a role in the contraction observed.⁴⁸

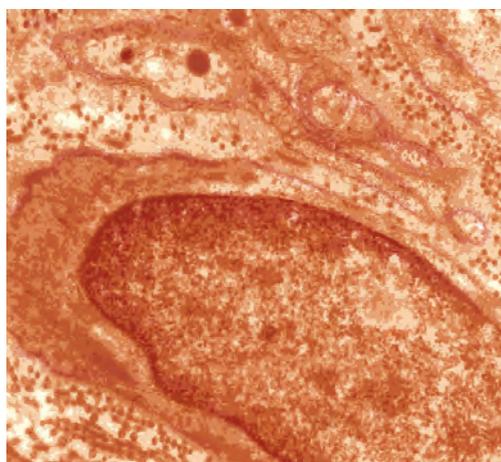


Figure 8: This is an electron photomicrograph of a typical smooth muscle-like cell within the fascia cruris. Above it may be seen the terminal portion of a type IV (unmyelinated) sensory neuron. *Photo courtesy of J. Staubesand. Used by permission.*

A few years later, in 1996, German anatomy professor Jochen Staubesand published an exciting new paper. He and his colleague Dr. Yi Li studied the fascia cruris in humans during a period several years, using electron photomicroscopy, and found **smooth muscle-like cells** embedded within this fascia’s collagen fibers (Fig. 8).⁵¹

Staubesand also described a rich intrafascial supply of sympathetic nerve tissue and sensory nerve endings. Based on his findings, he concluded that it is likely that these fascial smooth muscle cells enable the sympathetic nervous system to regulate a **fascial pre-tension** independent of the muscular tonus.^{52,53} He, therefore, postulated that this new understanding of fascia as an actively adapting organ gives fascia a much higher functional importance, and that the close links between fascia and autonomic may have far-reaching clinical implications. The palpatory stiffness of fascia could then be seen to be driven, at least sometimes, by strong sympathetic activity of the autonomic nervous system.

Fascial Contractions Caused by Multiple Factors

Inspired by the combined findings of Yahia and Staubesand, my research group at Ulm University conducted a histological examination of fascial tissues from different anatomical locations in 32 human donors, ages 17 to 91. It was demonstrated that **myofibroblasts** exist in all of these fascial tissues.⁵⁴ Myofibroblasts are known to have high contractile properties and can actively contract in a smooth muscle-like manner.^j

With in vitro examinations, we also explored the contractile capacity of rat lumbar fasciae in response to chemical stimulation. We discovered that substances associated with wound healing and inflammation – such as thromboxane and mepyramine – were able to trigger long-lasting tissue contractions.^k These contractile responses were large enough to predict a palpable tissue response when applied to the anatomical properties of the human body.

Does this mean that Staubesand's suggestion that fascial contraction is regulated by the sympathetic nervous system has little substance in reality? Not at all. Recent studies suggest that multiple factors can be responsible for fascial contractions and stiffness. For example, the work of pharmacology professor Mohammad H. Pipelzadeh suggests that alterations in the pH level of the cellular environment can impact myofibroblast contractility.⁵⁵

Pain can induce a shift in the pH level of the ground substance.⁵⁶ The same is true for nutrition, as well as for chronic hyperventilation.⁵⁷ Therefore, all these factors – in addition to mechanostimulation, chemical stimulation, and the state of the sympathetic nervous system – may influence the regulation of fascial stiffness.

How Stress Can Lead to Fascial Stiffness

Recent findings from the field of psychoneuroimmunology have uncovered additional confirmation of Staubesand's conclusion that sympathetic activation causes fascial contractions. A recent study by Sourojit Bhowmick, a researcher at Yale University School of Medicine, demonstrated that if you stimulate the sympathetic nervous system, it sends out the cytokine TGF-beta-1 to elicit an immune system response.^{58,1}

Interestingly, TGF-beta-1 is also known as the most reliable and most potent physiological stimulator of myofibroblast contraction. As shown by the work of James J. Tomasek, a professor in the department of cell biology at the University of Oklahoma College of Medicine, an

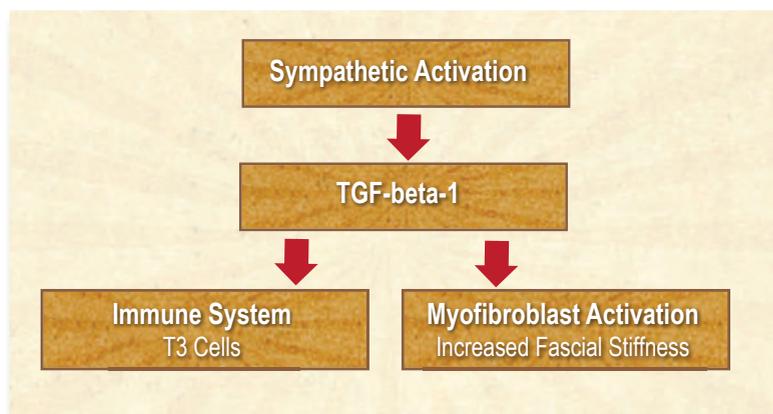


Figure 9: Here we see how stress can influence fascial stiffness. An increase of sympathetic activation leads to alterations of the immune system. A potent cytokine and well-known specific growth factor, TGF-beta-1, has been shown to serve as a bridge for that transmission. However, TGF-beta-1 is also known to increase tissue stiffness via activation of the contractile behavior of myofibroblasts.

^j Fascial myofibroblasts can be seen as a phenotype of fibroblast with very high contractile properties. It has long been known that fibroblasts often transform into **myofibroblasts**, which stress fiber bundles containing smooth muscle actin and can, therefore, actively contract in a smooth muscle-like manner. This happens in pathologies such as Dupuytren's contracture, liver cirrhosis, rheumatic arthritis, and frozen shoulder. However, it is also a productive element of early wound healing, and myofibroblasts are regularly found in healthy skin, as well as in the spleen, uterus, ovaries, circulatory vessels, periodontal ligaments, and pulmonary septa.³²

^k Interestingly, our original hypothesis – that autonomic nervous system neurotransmitters, such as adrenaline, noradrenaline, and acetylcholine, might be able to elicit contractile responses large enough to translate to a palpable tissue response – was thwarted.

increase in TGF-beta-1 can induce tissue contracture mediated by myofibroblasts.⁵⁹ These studies indicate that an increase in sympathetic activation, as caused by stress, can lead to increased fascial stiffness.

Figure 9 and Figure 10, therefore, illustrate our proposed connections between the sympathetic nervous system and fascial contractions.

How Manual Therapy Can Reduce Stiffness

Fortunately, although an increase in stress can cause fascial stiffness, a bodyworker can combat this stiffness through skillful manipulation of the fascial mechanoreceptors. Therapeutic manipulation of the

fascial mechanoreceptors – particularly the Ruffini endings and interstitial receptors – can reduce stiffness by signaling inhibition of the sympathetic nervous system.

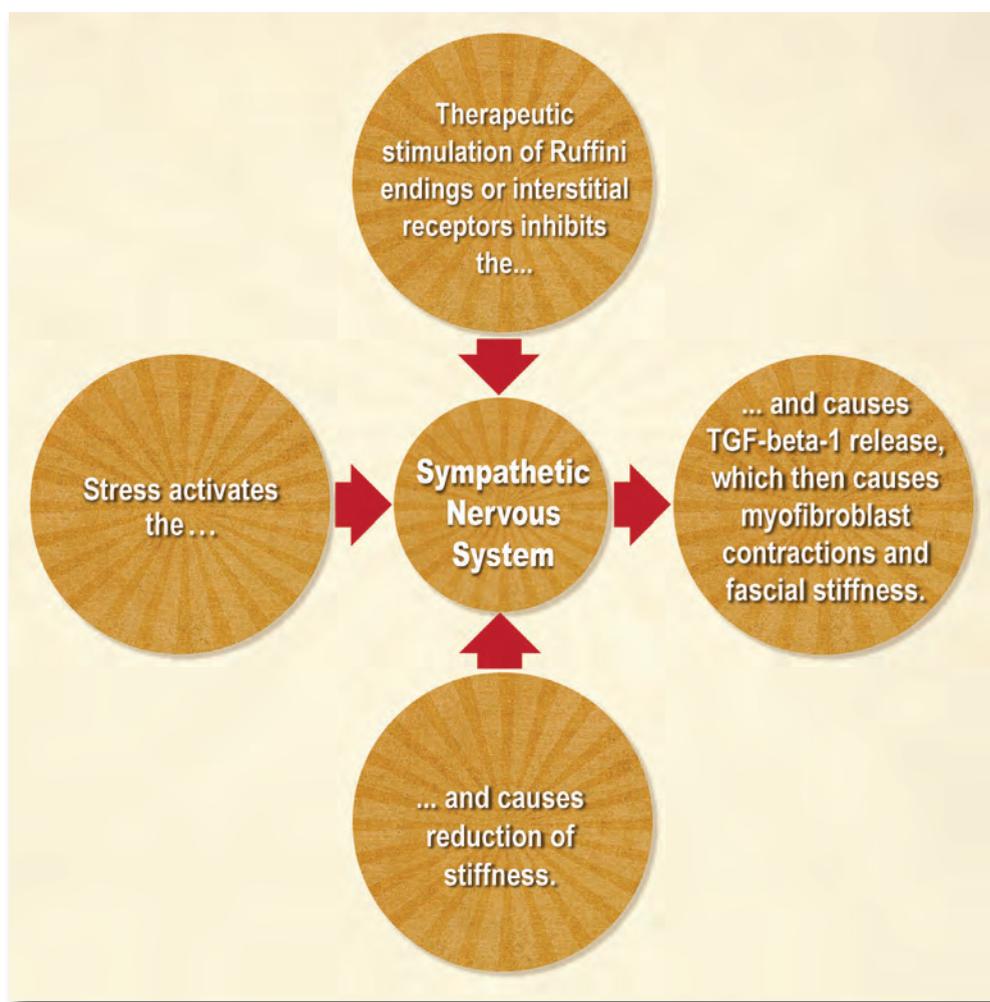


Figure 10: The circles above show the interactions between the sympathetic nervous system and fascia. An increase in sympathetic activation can increase fascial stiffness via the stimulatory action of TGF-beta-1 on fascial myofibroblasts. A therapeutic stimulation of fascial mechanoreceptors, particularly Ruffini endings or interstitial receptors, may reduce stiffness by inducing changes in the state of the sympathetic nervous system.

¹ While it has been known for some time that sympathetic activation leads to altered activity in the immune system, particularly to the deployment of regulatory T-cells in the body's lymph nodes, the exact transfer mechanism by which the sympathetic nervous system stimulates these cells remained enigmatic. A new study has elucidated the missing link in that transmission – it is TGF-beta-1, a well-known multipotent cellular growth factor in the human body.⁵⁸ This study demonstrates that an increase in sympathetic activation uses an increase in TGF-beta-1 to elicit the expression of specific T-regulatory cells in lymph nodes.

Acupuncture Meridians and Fascia

Electron photomicroscopy studies of the fascia cruris show there are numerous perforations of the superficial fascia layer, all characterized by a perforating triad of vein, artery, and nerve (Fig. 11). Staubesand could ascertain that most of the perforating nerves in these triads are unmyelinated sympathetic nerves.^{52,53}



Figure 11: The superficial fascia is perforated at specific points by a triad of nerve (left), vein (large body in middle), and artery. According to German anatomy professor Hartmut Heine, most of these perforation points are topographically identical with traditional Chinese acupuncture points.

A study by German anatomy professor Hartmut Heine also documented the existence of these triad perforation points in the superficial fascia. Heine found that the majority – 82 percent – of these perforation points are topographically identical with traditional Chinese acupuncture points.⁶⁰

This discovery prompted German surgeon Dr. Johann Bauer to conduct a clinical study with Heine. When looking at these fascial perforation points in patients suffering from chronic shoulder-neck or shoulder-arm pain, Bauer and Heine found that the perforation points in these patients showed a peculiar anomaly. The perforating vessels were “strangled” together by an unusually thick **ring of collagen fibers**, directly on top of the perforation hole. Bauer

treated these points with microsurgery to loosen the strangulations and achieve a **freer exit** of those vessels. This resulted in a significant improvement for the patients.⁶¹

While many took this as clear evidence of a new mechanical explanatory model for pain in relation to acupuncture points, only a year later, Francisco M. Kovacs, a back pain researcher from Spain, published a study that seems to question some of Bauer and Heine’s assumptions – and adds an exciting new dimension.⁶² Using a well-orchestrated double-blind study design, surgical staples were implanted under the skin of patients suffering from chronic low back pain. An interesting point is that the location of the implants was defined by their segmental innervation, and was carefully chosen **not** to coincide with Chinese acupuncture points. The result: Kovacs’ treatment led to a clear reduction of pain in the majority of his patients, with improvements at least statistically similar to those reported in the patients of Bauer and Heine.

Kovacs suggested the following explanation: Most likely, a class of liquid neuropeptides called enkephalins is released by both treatments. The enkephalins then counteract the release of substance P, bradykinin, and other neuropeptides associated with pain, which support the activation of nociceptive fibers. In other words, the stimulation of certain nociceptors and/or mechanoreceptors beneath the skin stimulates the release of specific neuropeptides that help depolarize already activated pain receptors that have been instrumental in the maintenance of chronic pain.⁶²

A Dynamic Systems Approach

The beauty of Kovacs' approach is his inclusion of the liquid aspects of the nervous system, his view of the nervous system as "a wet tropical jungle." Compared to the more mechanically-oriented treatment approach of Bauer and Heine, Kovacs sees the body as a **cybernetic system**, in which an intervention stimulates complex internal self-regulatory processes.

Cybernetic approaches can often be represented as flow charts, which visually simplify complex

dynamic interdependencies. Figure 12 can be seen as a first attempt toward an analysis of some of the neural factors behind immediate fascial plasticity. It includes several of the different feedback loops described earlier in this chapter. This flow chart does not include any neuroendocrine aspects, although it is very likely that they are significantly involved in myofascial manipulation.

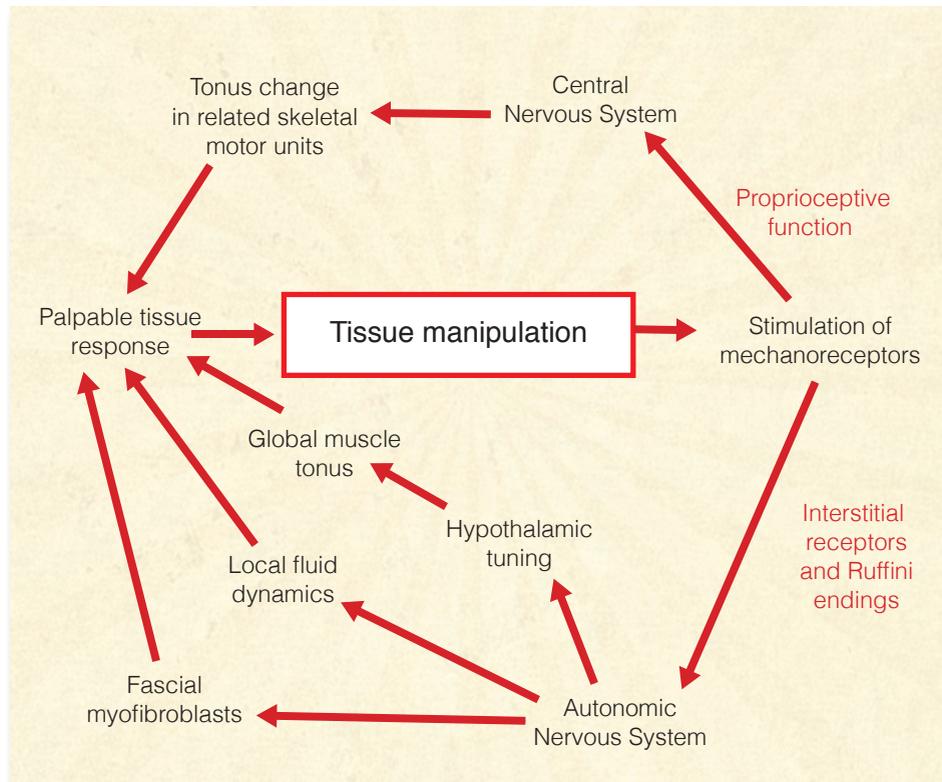


Figure 12: This flow chart outlines several processes involved in the neural dynamics of immediate tissue plasticity in myofascial manipulation. The chart includes the four different feedback loops discussed earlier in this chapter. The practitioner's manipulation stimulates intrafascial mechanoreceptors, which are then processed by the central nervous system and the autonomic nervous system. The response of the central nervous system changes the tonus of some related striated muscle fibers. The autonomic nervous system response includes an altered global muscle tonus, a change in local vasodilation and tissue viscosity, and a lowered tonus of intrafascial smooth muscle cells.

Shifting Models: From Hero Technician to Humble Midwife

It seems clear that, in order to better understand and use fascial plasticity, we need to include the self-regulatory dynamics of the nervous system. This means a shift in the attitude of the practitioner. If we are

willing to move from a mechanical view of the body to one that includes the neuroendocrine system, we do well to train ourselves to think more in terms of nonlinear system dynamics.

<i>Table 2</i> Classical Concept: THE BODY AS A MECHANICAL OBJECT	New Neurobiological Model: THE BODY AS A SELF-REGULATORY PROCESS
The body is seen as a perfect or imperfect machine , governed mainly by classical Newtonian physics.	The body is seen as a self-regulating (SR) biological organism , involving nonlinear system dynamics, complexity and autopoiesis.
Typical " industrial age " viewpoint	Typical " information age " viewpoint
Clear distinction between structure and function	No clear distinction between structure and function
Less focus on nervous system	Strong inclusion of nervous system
Subject-object separation ("principles of intervention ")	Subject-object connection (" interaction " instead of intervention)
Problem - solving attitude	Focus on enhancing already existing self regulation
A machine has a limited number of variables. An inner sense of absolute certainty in the practitioner is therefore seen as achievable and as desirable.	The system has high degree of complexity with almost unlimited variables. Practitioner personality needs to be comfortable operating with uncertainty principles.
Local " precision " is important and admired	Good timing and gradation (dosage) are becoming at least as important
"Master Technician" as idol	"Facilitator" or "Midwife" as idols
Typical work example: Direct mobilization of a precise " spinal fixation " or sacral torsion by the practitioner.	Typical work example: Inclusion of facilitated active client micromovements during the hands-on work.

The self-regulatory complexity of the nervous system can be compared with that of a rainforest or a metropolitan city. According to Peter Senge, founder of the Center for Organizational Learning at MIT's Sloan School of Management, when dealing with such complex systems, it usually does not work well to assume the role of an expert or **master** – heroically intervening from the outside – who believes he can predict the results of his intervention with certainty. Such an approach often yields positive short-term effects, but these kinds of linear interventions can lend themselves to producing unforeseen and detrimental long-term reactions.⁶³

Usually, it works better to assume the more humble role of *facilitator*, who is curiously interested in

learning and whose personality is more comfortable dealing with uncertainty principles. In the context of a bodywork session, practitioner and client then work together as a “learning team.”⁶⁴

Table 2 shows some of the consequences of this shift. It is a move away from seeing practitioner and client as clearly separable entities (subject and object) and from discussing different “principles of intervention” in manual therapy, in which the practitioner performs a number of skilled techniques on a mostly passive client. Instead, it is suggested that there is benefit to be gained by involving the client as an active partner in an interaction process – for example, with specific micromovements during the fascial manipulations.



Figure 13: A door blocked by a rock requires a different approach than a door blocked by an animated obstacle. Similarly, a blocked joint or immobile tissue can be understood in purely mechanical terms or as an active self-regulatory system. The choice of approach depends largely on whether the practitioner sees any neural dynamics involved in the specific situation on the client's side. This chapter makes a point of seeing fascia as innervated and alive, suggesting it should be treated more with the second approach. Illustration by Twyla Weixl, Munich, Germany.

Note that the common distinction between structure – bones, dense fibrous connective tissue – and function – neuromuscular organization – is no longer useful within this new picture. Nobel laureate Ludwig von Bertalanffy puts it this way:

The antithesis of structure and function, morphology and physiology, is based upon a static conception of the organism. In a machine, there is a fixed arrangement that can be set in motion but can also be at rest. In a similar way, the pre-established structure of, say, the heart is distinguished from its function, namely, rhythmical contraction. Actually, this separation between a pre-established structure and processes occurring in that structure does not apply to the living organism. For the organism is the expression of an everlasting, orderly process, though, on the other hand, this process is sustained by underlying structures and organized forms. What is described in morphology as organic forms and structures is in reality a momentary cross section through a spatio-temporal pattern.

What are called structures are slow processes of long duration, functions are quick processes of short duration. If we say that a function such as the contraction of a muscle is performed by a structure, it means that a quick and short process is superimposed on a long-lasting and slowly running wave.⁶⁵

The role of a “Master Technician” in Table 2 can best be described by the following story. The heating system of a big steamboat was broken and for

several days, and nobody could fix it. Finally, a master technician was called in. He just walked around and looked at everything, then he took out a little hammer from his pocket and hit a little valve, which immediately fixed the problem, and the machine started working again. When his bill of \$1,000 arrived, the captain didn’t want to believe there could be such a high sum for so little work. He asked for an itemized bill, and the next day, the new bill arrived. It said:

For adjusting a little valve: \$ 0.01

For knowing where: \$ 999.99

Many bodywork practitioners still worship this story as an ideal of *mastery* in their work, although it clearly belongs in the realm of dealing with a mechanical universe. If one is willing to deal with fascia in a dynamic systems perspective, it is more appropriate to assume the role of a *midwife* or *facilitator* who is skillfully assisting a self-regulatory process of the organism. This ideal is expressed in the Chinese saying:

Give a man a fish, and you feed him for a day.

Teach him how to fish, and you feed him for a lifetime.

Active Client Participation

A groundbreaking study by Dr. Lorimer Moseley, professor of clinical neurosciences and director of the Body in Mind research group, demonstrated that skillful mechanostimulation on the hand can be a very effective treatment for complex regional pain syndrome. However, the effectiveness of the treatment depends on the cortical attention of the patient. If the patients were asked to accompany each touch with detailed attention, via a perceptual discrimination task, the treatment was highly effective. The exact same type of mechanostimulation showed zero effectiveness when the patients were allowed to read the newspaper and were not required to give detailed attention to each stimulation.⁶⁶

The proposed mechanism for the effectiveness of the treatment included a remapping of the respec-

tive body part representation in the somatomotor cortex. While such cortical remapping may not have the same importance in all other musculoskeletal dysfunctions, most body therapists would probably agree to the notion that the ultimate sustainability question – *How long do the session changes last?* – depends to a large degree on how much the client “owns” or “embodies” the recently gained relationships within his or her internal body schema and psychological body image.

How can we engage the client to pay curious attention to each detail of our fascial manipulation? Working at the edge of pain works well with most clients. The same goes for the perception and projection of charisma and status

on the practitioner, for an atmosphere of magic, or the setting of appropriately timed pauses after each intervention.

Another tool, which is even more reliable and also easily teachable, is the engagement of the client's participation with active micromovements during the hands-on work. Figure 14 shows a typical example of using active micromovement participation in a

sitting client. Refined verbal and tactile guidance from the practitioner help facilitate the subtle slow-motion participation of the client, such that the nervous system is more deeply involved in the coordination around a specific joint or area. Inspired by the Continuum Movement work of Emilie Conrad and Susan Harper, the smaller and slower the movements are orchestrated, the more nurturing the perceptual involvement becomes.⁶⁷

Figure 14: This photo shows an example of the use of active movement participations (AMPs) of the client in a Rolwing® structural integration session. While deeply melting with one hand into the tissue and specific joints of the upper thorax, the author guides the client to support his myofascial work with subtle and non-habitual slow motion participations. Here, the client performs a lateral bending movement of the thorax, combined with a cranially directed extension (following the elbow), in order to increase an opening of the thoracic vertebral joints. Photo courtesy of the European Rolwing Association. Used by permission.



Conclusion

Fascia is alive. The practitioner working with fascial tissue should understand that it is innervated by four different kinds of mechanoreceptors. Without an inclusion of the responsiveness of these mechanoreceptors to various kinds of touch, the immediate effects of tissue release in myofascial manipulation cannot be adequately explained.

Manual stimulation of these sensory endings probably leads to tonus changes in the motor units that are mechanically linked to the tissue beneath the practitioner's hand. At least some of these responses are primarily regulated by a change in gamma motor tone.

Of particular interest are the Ruffini organs, with their high responsiveness to tangential pressure, and the very rich network of interstitial receptors. Stimulation of both these receptors can trigger profound changes in the autonomic nervous system.

There are strong links between fascia and the autonomic nervous system that affect fascial tonus and local tissue viscosity. Therefore, a shift from a mechanically-oriented "technician" point of view toward an inclusion of the self-regulation dynamics of the client's nervous system is advocated.

References

- Varela, F.J., & Frenk, S. (1987). The organ of form: towards a theory of biological shape. *J Social Biol Struct*, 10, 73-83.
- Barnes J.F. (1990). *Myofascial Release: The Search for Excellence*. Paoli, PA: Rehabilitation Services Inc.
- Cantu, R.I., & Grodin, A.J. (1992). *Myofascial Manipulation: Theory and Clinical Application*. Gaithersburg, MD: Aspen Publishers.
- Chaitow, L. (1980). *Soft-Tissue Manipulation*. Rochester, VT: Healing Arts Press.
- Paoletti, S. (1998). *Les fascias – Role des tissues dans la mecanique humaine*. Vannes cedex, France: Le Prisme.
- Rolf, I.P. (1977). *Rolfing: The Integration of Human Structures*. Santa Monica, CA: Dennis-Landman.
- Ward, R.C. (1993). Myofascial Release Concepts. In J.V. Basmajian and R.E. Nyberg (Eds.), *Rational Manual Therapies*. Baltimore, MD: Williams & Wilkins.
- Juhan, D. (1987). *Job's Body: A Handbook for Bodywork*. Barrytown, NY: Station Hill Press.
- Twomey, L., & Taylor, J. (1982). Flexion, creep, dysfunction and hysteresis in the lumbar vertebral column. *Spine*, 7(2), 116-122.
- Currier, D.P., & Nelson, R.M. (1992). *Dynamics of Human Biologic Tissues*. Philadelphia, PA: F.A. Davis Company.
- Oschman, J.L. (2000). *Energy Medicine*. Edinburgh, United Kingdom: Churchill Livingstone.
- Athenstaedt, H. (1974). Pyroelectric and piezoelectric properties of vertebrates. *Ann NY Acad Sci*, 238, 68-110.
- Juhan, D. (1998). *Job's Body: A Handbook for Bodywork* (3rd ed.). Barrytown, NY: Station Hill Press.
- Threlkeld, A.J. (1992). The Effects of Manual Therapy on Connective Tissue. *Phys Ther*, 72(12), 893-901.
- Chaudhry, H., Schleip, R., Zhiming, J., Bukiet, B., Maney, M., & Findley, T. (2008). Three-Dimensional Mathematical Model for Deformation of Human Fasciae in Manual Therapy. *J Am Osteopath Assoc*, 108(8), 379-390.
- Schleip, R. (1989). A new explanation of the effect of Rolfing. *Rolf Lines*, 15(1), 18-20.
- Still, A.T. (1899). *Philosophy of Osteopathy*. Kirksville, MO: Academy of Osteopathy.
- Kandel, E.R. (1995). *Essentials of neural science and behavior*. New York, NY: Appleton & Lange.
- Schleip, R. (2000). Lichtblicke im Dschungel der Gehirnforschung. *FeldenkraisZEIT*, 1, 47-56.
- Engeln, H. (1994). Konzert der Muskeln und Sinne. *GEO Wissen*, 1(Mai), 90-97.
- Folkow, B., Gelin, L.E., Lindell, S.E., Stenberg, K., & Thoren, O. (1962). Cardiovascular Reactions During Abdominal Surgery. *Ann Surg*, 156(6), 905-913.
- Koizumi, K., & Brooks, C.M. (1972). The integration of autonomic system reactions: a discussion of autonomic reflexes, their control and their association with somatic reactions. *Ergeb Physiol*, 67, 1-68.
- Gellhorn, E. (1967). *Principles of Autonomic-Somatic Integrations: Physiological Basis and Psychological and Clinical Implications*. Minneapolis, MN: University of Minnesota Press.

24. Arbuckle, B.E. (1994). *Selected Writings*. Indianapolis, IN: American Academy of Osteopathy.
25. Gershon, M.D. (1999). *The Second Brain*. New York, NY: Harper-Collins.
26. Cottingham, J.T. (1985). *Healing Through Touch: A History and a Review of the Physiological Evidence*. Boulder, CO: Rolf Institute.
27. Jami, L. (1992). Golgi tendon organs in mammalian skeletal muscle: functional properties and central actions. *Physiol Rev*, 73(3), 623-666.
28. Lederman, E. (1997). *Fundamentals of Manual Therapy*. Edinburgh, United Kingdom: Churchill Livingstone.
29. Huijing, P.A. (2009). Epimuscular myofascial force transmission: a historical review and implications for new research. International Society of Biomechanics Muybridge Award Lecture, Taipei, 2007. *J Biomech*, 42(1), 9-21.
30. Burke, D., & Gandeva, S.C. (1990). Peripheral Motor System. In G. Paxinos (Ed.), *The Human Nervous System* (p. 133). San Diego, CA: Academic Press.
31. Stilwell, D. (1957). Regional variations in the innervation of deep fasciae and aponeuroses. *Anat Record*, 127(4), 635-653.
32. van den Berg, F., & Cabri, J. (1999). *Angewandte Physiologie – Das Bindegewebe des Bewegungsapparates verstehen und beeinflussen*. Stuttgart, Germany: Georg Thieme Verlag.
33. Yahia, L., Rhalmi, S., Newman, N., & Isler, M. (1992). Sensory innervation of human thoracolumbar fascia: An immunohistochemical study. *Acta Orthop Scand*, 63(2), 195-197.
34. Hanna, T. (1998). *Somatics: Reawakening the Mind's Control of Movement, Flexibility, and Health*. Cambridge, MA: Da Capo Press.
35. Kruger, L. (1987). Cutaneous Sensory System. In G. Adelman (Ed.), *Encyclopedia of Neuroscience* (p. 293). Boston, MA: Birkhäuser.
36. Mitchell, J.H., & Schmidt, R.F. (1983). Cardiovascular Reflex Control by Afferent Fibers from Skeletal Muscle Receptors. In J.T. Shepherd et al (Eds.), *Handbook of Physiology, The Cardiovascular System, Peripheral Circulation and Organ Blood Flow* (pp. 623-658). Bethesda, MD: American Physiological Society.
37. Sakada, S. (1974). Mechanoreceptors in fascia, periosteum and periodontal ligament. *Bull Tokyo Med Dent Univ*, 21, S11-13.
38. Coote, J.H., Pérez-González, J.F. (1970). The response of some sympathetic neurons to volleys in various afferent nerves. *J Physiol-London*, 208, 261-278.
39. Johansson, B. (1962). Circulatory response to stimulation of somatic afferents. *Acta Physiol Scand*, 62(S198), 1-92.
40. Eble, J.N. (1960). Patterns of response of the paravertebral musculature to visceral stimuli. *Am J Physiol*, 198, 429-433.
41. Schleip, R. (1993). Primary reflexes and structural typology. *Rolf Lines*, 21(3), 37-47.
42. Glaser, V. (1980). *Eutonie*. Heidelberg, Germany: Karl F. Haug Verlag.
43. Henatsch, H-D. (1976). Bauplan der peripheren und zentralen sensomotorischen Kontrollen. In *Physiologie des Menschen* (vol. 14). München, Germany: Urban and Schwarzenberg.
44. Johansson, H., Sjolander, P., & Sojka, P. (1991). Receptors in the knee joint ligaments and their role in the biomechanics of the joint. *Critical Rev Biomed Eng*, 18(5), 341-368.
45. Basmajian, J.V., & De Luca, C.J. (1985). *Muscles Alive: Their Functions Revealed by Electromyography*. Baltimore, MD: Williams & Wilkins.
46. Barnes, M.F. (1997). The Basic Science of Myofascial Release. *J Bodywork Move Ther*, 1(4), 231-238.
47. Pischinger, A. (1991). *Matrix and Matrix Regulation: Basis for a Holistic Theory in Medicine*. Brussels, Belgium: Haug International.
48. Yahia, L.H., Pigeon, P., & DesRosiers, E.A. (1993). Viscoelastic properties of the human lumbodorsal fascia. *J Biomed Eng*, 15(5), 425-429.
49. Chaitow, L., & DeLany, J.W. (2000). *Clinical Application of Neuromuscular Techniques, Volume 1*. Edinburgh, United Kingdom: Churchill Livingstone.
50. Price, J.M., Patitucci, P., & Fung, Y.C. (1981). *Biomechanics: Mechanical Properties of Living Tissues* (pp. 371-391). New York, NY: Springer Verlag.
51. Staubesand, J., & Li, Y. (1996). Zum Feinbau der Fascia cruris mit besonderer Berücksichtigung epi- und intrafasziärer Nerven. *Manuelle Medizin*, 34, 196-200.
52. Staubesand, J., & Li, Y. (1997). Begriff und Substrat der Faziensklerose bei chronisch-venöser Insuffizienz. *Phlebologie*, 26, 72-79.
53. Staubesand, J., et al. (1997). La structure fine de l'aponévrose jambière. *Phlebologie*, 50(1), 105-113.
54. Schleip, R., Lehmann-Horn, F., & Klingler, W. (2006). Fascia is able to contract in a smooth muscle-like manner and thereby influence musculoskeletal mechanics. In D. Liepsch (Ed.), *Proceedings of the 5th World Congress of Biomechanics, July 29 – August 4, 2006, Munich, Germany* (pp. 51-54). Pianoro, Italy: Medimond International Proceedings.
55. Pipelzadeh, M.H., & Naylor, I.L. (1998). The in vitro enhancement of rat myofibroblast contractility by alterations to the pH of the physiological solution. *Eur J Pharmacol*, 357(2-3), 257-259.

56. Steen, K.H., Steen, A.E., Kreysel, H.W., & Reeh, P.W. (1996). Inflammatory mediators potentiate pain induced by experimental tissue acidosis. *Pain*, 66(2-3): 163-70.
57. Chaitow, L., Bradley, D., & Gilbert, C. (2002) *Multi-disciplinary Approaches to Breathing Pattern Disorders*. Edinburgh, United Kingdom: Churchill Livingstone.
58. Bhowmick, S., Singh, A., Flavell, R.A., Clark, R.B., O'Rourke, J., & Cone, R.E. (2009). The sympathetic nervous system modulates CD4(+)FoxP3(+) regulatory T cells via a TGF-beta-dependent mechanism. *J Leukoc Biol*, 86(6), 1275-1283.
59. Tomasek, J.J., Gabbiani, G., Hinz, B., Chaponnier, C., & Brown, R.A. (2002). Myofibroblasts and mechano-regulation of connective tissue remodeling. *Nat Rev Mol Cell Biol*, 3, 349-363.
60. Heine, H. (1995). Functional anatomy of traditional Chinese acupuncture points. *Acta Anat*, 152, 293.
61. Bauer, J., & Heine, H. (1998). Akupunkturpunkte und Fibromyalgie – Möglichkeiten chirurgischer Intervention. *Biologische Medizin*, 6(12), 257-261.
62. Kovacs, F.M., Abaira, V., Pozo, F., Kleinbaum, D.G., Belrán, J., Mateo, I., ... Morillas, L. (1997). Local and Remote Sustained Trigger Point Therapy for Exacerbations of Chronic Low Back Pain: A Randomized, Double-Blind, Controlled, Multicenter Trial. *Spine*, 22(7), 786-797.
63. Senge, P.M. (1990). *The Fifth Discipline: The Art and Practice of The Learning Organization*. New York, NY: Doubleday.
64. Petersen, S. (2000) *Mana Integrative Therapies*. Manaia, New Zealand: Mana Publications.
65. von Bertalanffy, L. (1952). *Problems of Life*. New York, NY: Harper and Row.
66. Moseley, G.L., Zalucki, N.M., & Wiech, K. (2008). Tactile discrimination, but not tactile stimulation alone, reduces chronic limb pain. *Pain*, 137(3), 600-608.
67. Conrad, E. (2007). *Life on Land: The Story of Continuum*. Berkeley, CA: North Atlantic Books.



Robert Schleip, Ph.D.

Robert Schleip is the director of the Fascia Research Project, part of the division of neurophysiology at Ulm University in Germany. In 2007, he received the Vladimir Janda Award for Musculoskeletal Medicine for his pioneering work on active fascial contractility.

Schleip holds a doctorate in human biology and a master's in psychology. He has been a Certified Rolfer® since 1978 and currently serves as research director for the European Rolwing Association. Schleip is also a Rolwing instructor and Feldenkrais® teacher, and he has been teaching anatomy for more than 25 years.

For more information, visit www.fasciaresearch.de.



While deeply melting with one hand into the tissue and specific joints of the upper thorax, I guide the client to support myofascial work with subtle and non-habitual slow motion participations.